

Malabsorption Syndrome

Definition:

- Failure of absorption of one or more of the nutrient (fat, proteins, CHO, minerals..)
- **Steatorrhea** (fatty stool) remains the main mark of malabsorption.

Causes: 4

I- Gastric Causes: 4

- 1-Gastrectomy.
- 2-Atrophic gastritis.
- 3-Cancer stomach → achlorhydria (↓HCl)→Bacterial Contamination of intestine.
- 4-Zollinger-Ellison's syndrome → hyperchlorhydria (↑HCl) → inhibits pancreatic lipase.

II- Hepato-biliary Causes : 4

- 1- Liver Cirrhosis
- 2- Chronic hepatitis.
- 3- Biliary obstruction.
- 4- Biliary fistula.

III- Pancreatic Causes : 4

- 1- Chronic pancreatitis
- 2- Cystic fibrosis
- 3- Cancer head of pancreas.
- 4- Pancreatectomy.

IV- Intestinal Causes : (the most common)

A) Primary causes:

- 1- Tropical sprue: unknown etiology but may be:
 - Bacterial, viral , parasitic infection.
 - Folic acid deficiency.
- 2- Coeliac disease: (*Non-tropical sprue* , *Gluten sensitive enteropathy*)
 - autoimmune disorder in which there is an abnormal reaction to gluten (protein found in wheat) → Ag. Ab reaction → damage of intestinal mucosa.
 - C/P : severe malabsorption syndrome , associated autoimmune diseases.
 - Complications : small intestine carcinoma , ↑incidence of lymphoma.
 - Investigations : Jejunal biopsy → villous atrophy , IgA antigliadin antibodies
 - Treatment : The only effective treatment is lifelong gluten free diet.

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B) Secodary to intestinal diseases:

- 1- **Short gut syndrome**: extensive intestinal resection → ↓ absorptive surface.
- 2- **Stagnant (blind) loop syndrome**: Stagnation of intestinal content e.g. strictures of small intestine , Diverticulosis → **bacterial overgrowth** → mucosal injury & nutrients utilization .
- 3- **Systemic diseases**: DM , amyloidosis, hypothyroidism , CHF.
- 4- **Infection**: -Bacterial over growth.
-TB enteritis, Giardia, Strongeloides.
- 5- **Inflammation**: Crohn's disease , Irradiation.
- 6- **Iatrogenic**: **A**ntacids , **B**iguanides , **C**holestyramine , **D**endivan .
- 7- **Lymphatic obstruction**:
 - Lymphoma.
 - Whipple's disease : (*mainly affects MAN*)
Malabsorption Syndrome, **A**rthritis , **N**europathy & lymphadenopathy.

Clinical picture:

4

I. General manifestations:

- | | |
|--------------------|-------------------------|
| 1- loss of weight. | 2- fatigue. |
| 3- fever | 4- clubbing of fingers. |

II. Intestinal manifestations:

- 1- **Steatorrhea**: Stool is **Pale**, bulky, offensive, floats on water, greasy, glistening.
- 2- **Diarrhea**: malabsorption usually results in diarrhea.
- 3- Audible intestinal sounds (*borborygmi*).
- 4- Distension, colic.

III. Nutritional deficiency:

- protein → muscle wasting, edema, recurrent infections
- Fat → loss of weight.
- CHO → hypoglycemia.

- Minerals:
 - Iron → anemia.
 - Na → Muscle cramps, hypotension.
 - K → arrhythmia.
 - Ca, Mg → tetany.
 - Iodine → Goitre.
- Vitamins:
 - A → night blindness, follicular hyperkeratosis.
 - D → Rickets, osteomalacia.
 - E → infertility.
 - K → Bleeding tendency.
 - C → Scurvy
 - B1 → Beri – Beri.
 - B2 → glossitis – gastritis.
 - B3 → **Diarrhea, Dermatitis, Dementia.**
 - B6 → peripheral neuritis.
 - B12 → Megaloblastic anemia.

IV. **C/P of the cause:** Coeliac disease , history of surgical operation

Investigation: 4

I- Biochemical investigation: 4

- 1- Estimation of fecal fat: (n : 6 gm/d)
 - in steatorrhea → total fat is increased (> 6 gm/d)
 - split fat → intestinal disease ▪ Non split fat → pancreatic disease
- 2- Pancreatic function tests.
- 3- Sugar curve : flat curve.
- 4- **D-xylose test:**

25 gm D xylose (*poorly metabolized sugar*) is given orally then collect urine over the next 5 hours.

 - Normally : 5 hours urine should contain at least 5 gm *provided that normal renal functions.*
 - In malabsorption syndrome : 5 hours will contain less than 5 gm

II- Hematological Investigations : **4**

- 1- **Blood picture:** Anemia. (Microcytic anemia due to iron deficiency or macrocytic anemia due to vitamin B₁₂ or Folate malabsorption.
- 2- **Plasma proteins :** hypoproteinemia.
- 3- **Serum electrolytes.**
- 4- **Prothrombin time:** may prolonged because of malabsorption of vitamin K.

III- Radiological investigations: **4**

- 1- CT 2- MRI 3- U.S.
- 4- Barium meals:
 - ◇ Dilatation of intestinal lumen. ◇ Loss of normal feathery appearance.

IV- Investigations for bacterial overgrowth :

1) 14 C- Xylose breath test :

- *14 C-Xylose is given orally then measure the 14 CO₂ in the breath.*
- In bacterial overgrowth : ↑ 14 CO₂ in the breath because more bacteria will act on 14 C- Xylose.

2) Aspiration of jejunal content then culture.

Treatment:

- 1- Treatment of the cause:
 - T.B enteritis : anti TB drugs.
 - Tropical sprue : antibiotic (tetracycline) & folic acid.
 - Coeliac disease :
 - The only effective treatment is lifelong gluten free diet.
 - cortisone.
- 2- Diet: Low fat, low fibers, non irritant diet.
- 3- Parenteral vitamins, minerals, fluid.
- 4- Symptomatic treatment :
 - e.g. Anti diarrheal drugs : Difenoxylate (*Lomotil*) , Loperamide (*Loperazin*).

Diarrhea

Definition:

It means an increase of stool liquidity , quantity (N: 200g/d) or frequency (N: 4 motions/d).

Etiology:

I- Etiology of acute diarrhea :

1- Infection:

- **Bacterial** : **S**almonella , **S**higella , E.**c**oli , **C**holera
- **Viral** : Rota virus , Norwalk virus
- **Protozoa**: E.histolytica , Malaria , Giardiasis
- **Helminthes**: Ascaris , strongyloids stercoralis.

2- Iatrogenic:

- * Laxatives * Antibiotic * Chemotherapy
- * Parasympathetic * Mg containing antacid. * allopurinol

3- Toxins:

- * Bacterial: staph, E.coli * Lead * arsenic

4- Diet:

- * Unripe fruit * Alcohol * Mushroom. * food allergy

5- **Nervous**: psychological stress e.g. before *internal medicine* exam. ☠

II. Etiology of chronic diarrhea:

1- **malabsorption syndrome**. (*enumerate its causes*)

2- Diseases of the colon :

- Irritable bowel syndrome.
- Inflammatory bowel diseases e.g. ulcerative colitis.
- Cancer colon.

3- Endocrinal causes :

- Thyrotoxicosis.
- Diabetic neuropathy.
- Addison's disease.
- Zollinger Ellison syndrome.

Mechanisms of Diarrhea:

- i. **Osmotic diarrhea:** Presence of non absorbed , hypertonic substances in intestinal lumen maintain fluid & prevent absorption (e.g. lactulose, sorbitol, magnesium).
- ii- **Secretory diarrhea** (*watery diarrhea*) : ↑↑ secretion of water & electrolytes into the lumen.
e.g. cholera , E.coli , ↑ VIPs (*vaso - active intestinal peptides*)
- iii- **Abnormality of intestinal motility** : e.g. Thyrotoxicosis, IBS, D neuropathy.
- iv- **Abnormality of intestinal mucosa** : e.g. inflammation.

Complications :

- ↓ H₂O → dehydration.
- ↓ K → hypokalemia.
- ↓ HCO₃ → Acidosis.

Diagnosis: *how to reach diagnosis of a case of diarrhea?*

1- **History analysis:**

- **Frequency:** >4/d → diarrhea
 - If the stool is of large volume & not excessive frequent → small bowel disease.
 - If the stool is of small volume & excessive frequent → large bowel disease.
- **Consistency:** - watery: inflammation
- greasy: malabsorption.
- **Color of stool:** - bloody as in ulcerative colitis.
- pale as in steatorrhea. - tarry as in melena.
- **Relation to meal:**
 - Osmotic diarrhea : Stops with fasting.
 - Secretory diarrhea : Continues with fasting.
- **Associated symptoms:**
 - **Abdominal pain:** all causes of diarrhea except drug induced & Thyrotoxicosis.
 - **Nausea & vomiting:** Acute infections.
 - **Fever:** infection, inflammation.
 - **Constipation:** IBS.

2- **Examination:**

- abdominal tenderness
- bowel sounds
- degree of general hydration
- examination per rectum: to exclude rectal mass or blood.

3- **Investigations:** 4 S

Not every patient who presents with diarrhea needs to be evaluated with these expensive tests, watchful waiting & symptomatic therapy with oral fluid are very enough.

- **Stool examination:**
 - for ova, cysts & parasites.
 - stool osmolarity
 - fat assay.
- **Sigmoidoscopy:** if a large bowel cause is suspected.
- **Small bowel radiology:** if a small bowel cause is suspected.
- **Serological tests.**

Plus

- **Investigations Of malabsorption syndrome :** (see before).

Treatment : 3 S

- **Specific :** treatment of the cause.
- **Symptomatic:**
 - Anti diarrhea : Diphenoxylate (*lomotil*) , Loperamide (*loperazine*)
 - Anti emetic: motilium
- **Supportive:**
 - Diet: ↓ fat, ↓ irritants , light diet.
 - Treatment of complications: Fluid – K – HCO₃.

DYSENTERY

Definition :

Diarrhea + Tenesmus + Blood + mucous in stool.

Tenesmus : *painful defecation with sense of incomplete bowel evacuation.*

Etiology :

- Amoebic dysentery
- Bacillary dysentery, Bilharzial dysentery.
- Cancer colon, rectum.
- Diverticulosis.
- Malaria
- Renal failure
- Giardia (v. rare).

Dysentery Scheme :

- Causative organism :
- Mode of infection :
- C/P : * Asymptomatic * Symptomatic * Complications
- Investigations : 4 S
- Treatment : 3 S

Amoebic dysentery

- **Causative organism** : *E. histolytica* (cyst form)

- **Mode of infection** : feco-oral

(*ingestion of cysts which resist gastric acidity → transformed into vegetative form in the intestine & then passes to the colon*)

- **Clinical picture** :

1- **Asymptomatic** : just cysts in the stool.

2- **Symptomatic** :

a. Acute.

b. chronic

a. **Acute Amoebic dysentery :**

- **Symptom:** *One word*

dysentery : (mild diarrhea (8/d) , Tenesmus , blood & mucous in stool)

- **Signs:**

○ **Local :** Tenderness in colon especially caecum & sigmoid.

○ **General :** - **No** fever (*superficial lesion*)

- **No** dehydration (*mild diarrhea*)

b. **Chronic Amoebic dysentery :**

- Recurrent attacks of acute dysentery alternating with normal bowel habits.

- Abdominal pain may occur (over the caecum , appendix , transverse colon).

3- Complications:

○ **Local:** Amoeboma (*mass in the right iliac fossa*)

○ **Systemic:** Amoebic hepatitis (*see hepatology*).

Investigations: 4S

- **Stool examination:** cysts , mucous , blood.

- **Sigmoidoscopy:** flask shaped ulcer with intervening healthy mucosa.

- **Small bowel radiology**

- **Serological tests :** antibodies may be detected in the serum of the patient

Treatment : 3 S

- **Specific :** *anti amoebic*

- **Symptomatic**

- **Supportive**

} **Like diarrhea**

Antiamoebic drugs:

4 , 3 , 2

I- **Luminal amoebicidal :** used for treatment of cysts (*chronic amoeba*)

1- Halogenated quinoline e.g. *di-Iodo-hydroxy quinoline*

- SE: Neuropathy, goiter - Dose: 500 mg t.d.s for 2 weeks.

2- Phenanthroline quinines (*Entobex*) : 500 mg t.d.s for 2 weeks.

3- Diloxanide (*Furamide*) : 500 mg t.d.s for 1 week

4- Antibiotic: paromomycin & erythromycin are directly amoebicidal.

II- Systemic drugs: (*Acute & extra intestinal Manifestation*)

Have no effect on cyst in intestinal lumen

- 1- Emetine: SE: Cardiotoxicity
- 2- Dehydro-emetine: less toxic
- 3- Chloroquine: used in hepatic amoebiasis.

III- Luminal & systemic (Mixed):

1- Metronidazole (*Flagyl*)

- Dose: 750 mg t.d.s for 10 days - SE: nausea, metallic taste.

2- Tinidazole: (*fasigyn*) : 2 gm daily for 5 days

Bacillary dysentery

Causative organism: G-ve bacilli (shigella)

Mode of infection: feaco-oral.

Clinical picture :

Asymptomatic

Symptomatic:

- 1st phase (1-2 days) : fever – colic – diarrhea.
- 2nd phase (1-2 weeks) :
 - Dysentery :
(severe watery diarrhea (15 /d), tenesmus , mucous & blood in stool)
 - **Abdominal pain** & tenderness.
 - **General signs:** fever, malaise , dehydration.

Complication:

urethritis – arthritis – iridocyclitis – UTI (**Reiter's syndrome**)

Investigation: 4S

- **Stool examination:** Excess WBCs & RBCs , Culture: shigella
- **Sigmoidoscopy:** diffuse inflammation with dirty yellowish pseudomembrane.
- **Small bowel radiology.**
- **Serological test.**

Treatment : 3 S

➤ **S**pecific :

- **Ciprofloxacin** : 500 mg twice daily for 5 days.
- Tetracycline: 2.5 gm in single oral dose.
- Septrin (*Co. Trimexazole*) : 2 tab twice daily for 5 days.

➤ **S**ymptomatic :

- Anti spasmodic : for severe abdominal pain.
- Anti diarrheal drugs : are **better to be avoided** as they decrease the intestinal motility and hence decrease the wash of the organisms.

➤ **S**upportive : ↓ irritants , Excess fluids, vitamins.

Bilharzial dysentery

Causative organisms:

Mainly due to *S. mansoni* & very rare due to *S. hematobium*.

Mode of infection:

The parasite penetrates the skin during swimming in infected water.

Clinical picture: Incubation period : 2 months

Asymptomatic

Symptomatic:

- (1) **Dysentery** : → Severe diarrhea alternating with constipation.
 → Tenesmus
 → Mucous & blood in stool

(2) **Bleeding per rectum**

(3) **Pericolic tender mobile mass in left iliac fossa (*bilharzioma*)**

(4) **Hepato-splenomegaly (*Egyptian splenomegaly*) .**

Complications:

- **Hepatic bilharziasis. (.....)** *see Hepatology*
- **Bilharzial cor-pulmonale (.....)**

Investigations: 3S + 3 B

- **S**tool analysis: Bilharzial ova
- **S**igmoidoscopy: polyps or ulcers mainly in recto-sigmoid area.
- **S**erological test (by ELISA) : Schistosoma antibodies.
- **B**arium enema: polyps.
- **B**lood picture: Anemia – esinophilia.
- Investigations for **p**ortal hypertension.

Treatment:**i. Anti bilharzial drugs:**

- ✎ **Praziquantel** (*Biltricid*)
- ✎ **Oxamniquine** (*vansil*) :20 mg/kg orally for 3 doses (against *S. mansoni* only)
- ✎ Niridazole (*ambilhar*) : 25 mg/kg/d orally for 7 days.
- ✎ Metrifonate : 10 mg/kg orally.(*S. hematobium*)
- ✎ Mirazid (*Commiphora ext.*): Herbal drug , dose : 12 capsules over 6 days.

Praziquantel :

- Mechanism of action : Ca influx into the worm → marked contraction & spastic paralysis of the worm.
- Dose: 40mg / kg in two divided dose.
- S/E: headache, abdominal pain , nausea, vomiting, pruritis, fever, elevation of liver enzymes.

NB :

- Antibilharzial drugs must be given early before tissue fibrosis.
- Repeated courses may be needed as the worms migrate into the liver & return back to the periphery
- Fever & vitamins deficiencies must be treated before starting therapy.

ii. Endoscopic polypectomy.**iii. Treatment of the complications e.g. Portal hypertension.****iv. Symptomatic & supportive treatment.**

VOMITING

Definition:

forceful expulsion of gastric content into the mouth.

Mechanism of vomiting reflex:

→ stimulus → centre → response (vomiting)

- **stimulus:**
 - peripheral (*GIT*).
 - Central e.g.: ↑ ICT.
 - Metabolic e.g.: uremia.
- **centre :** vomiting centre in the medulla.
- **Response:** vomiting.
 - ◇ Pylorus is closed. ◇ cardia is opened.
 - ◇ contraction of the diaphragm& abdominal wall muscles.

Etiology:

I- Peripheral causes: (*GIT* causes)

- Gastritis - peptic ulcer – cancer stomach
- Hepatitis - pancreatitis - Appendicitis
- Cholecystitis - peritonitis
- pyloric obstruction – intestinal obstruction.

II- Central causes:

- Psychic : bad smell, sight.
- Anorexia nervosa.
- ↑ ICT: Tumor , Hemorrhage.
- Pain: migraine, MI, renal colic.

III- Metabolic causes:

- Renal failure. - Liver cell failure.
- Acidosis (DKA).
- Electrolyte: ↑ Ca, ↓ Na, ↓ or ↑K

IV- Iatrogenic: MAD

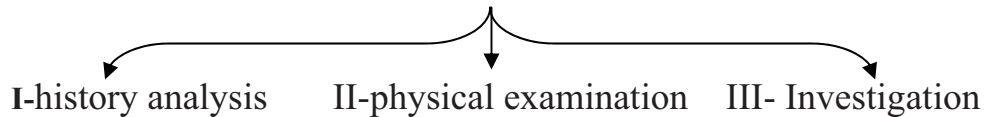
- morphine. - Alcohol. - Digitalis.

V- Pregnancy.

Complications of severe vomiting :

- dehydration.
- alkalosis (tetany).
- cerebral hemorrhage .
- pulmonary aspiration.

Diagnosis of a case of vomiting:



I- History analysis :

(1) Time of vomiting:

* Early morning :

- ☐ Pregnancy ☐ ↑ ICT ☐ Alcohol

* During meal : Esophageal cause.

* After meal by:

- ☐ ½ h : Gastric ulcer.
- ☐ 2-4 h : Duodenal ulcer.
- ☐ > 8h : Intestinal obstruction.

(2) Associated nausea: upper GIT causes.

- No nausea: ↑ ICT

(3) Associated Abdominal Pain : (GIT causes)

- e.g. peritonitis , intestinal obstruction , pancreatitis , cholecystitis
- painless vomiting : Neurological causes.
- pain relieved by vomiting : Gastric ulcer.

(4) Associated headache: ↑ ICT.

(5) History of drug intake : MAD

(6) Biliary symptoms:

Jaundice , Pruritise , Abdominal Pain , change of color of urine & stool.

(7) Frequency: **P**ersistent in **p**eritonitis.

II- Examination of vomiting:

- **Amount** : huge in pyloric obstruction .
- **Color** : coffee ground as in hematemesis.
- **Content** :
 - undigested food: pyloric obstruction.
 - bile: below ampulla of vater
 - blood: ulcer, cancer, Mallory weiss \$
 - F.B. : gall stone.
- **Odour**: offensive as in intestinal obstruction , malignancy.

III- Investigations: investigations for the causes.

Treatment :

- **Specific**: for the cause.
- **Symptomatic**:
 - ✍ Antiemetic : motilum , primpran.
 - ✍ Antidiarrheal drugs.
- **Supportive** : correct dehydration & electrolyte imbalance .

CONSTIPATION

Definition: Infrequent passage of hard stool .

Causes: 7 I

- i. **Immobility** (prolonged rest in bed).
- ii. **↓ Intake** of fluid & fibers .
- iii. **Irritable bowel syndrome** .
- iv. **↓ Intestinal motility** : hypothyroidism , DM , ↑ Ca , ↓ K .
- v. **Iatrogenic**: codeine , morphine , anticholinergic, aluminum hydroxide.
- vi. **Inflammation** : ulcerative colitis , diverticulitis .
- vii. **Intestinal obstruction** : cancer colon , stricture .

INFLAMMATORY BOWEL DISEASE

(Crohn's disease & ulcerative colitis)

Etiology:

- Is still questionable.
- Most probably autoimmune. (The local T cells are angry with something in the food)
- Genetic factor: play a great role.
- Infections (T.B, measles): not confirmed.
- Psychological factor may play a role.

	Crohn's disease	Ulcerative colitis
Pathology:		
- Site:	Affects any part of GIT from mouth to anus, esp. terminal ileum (70%).	- Limited to colon & rectum. - Terminal ileum can be affected in 5% only
- Layers	All three (<i>transmural</i>)	- Mucosa only
- Granuloma	Yes in (30%)	No
- Crypt abscesses	No	Yes (30%)
	N.B: finding of granuloma = crohn's disease	N.B: finding of crypt abscesses = ulcerative colitis
- Fibrosis	Server	No
- Pattern of colonic involvement	Skip lesions	Continuous involvement
C/P:		
- Sex	Equal	Slightly more in ♀ ☺
- Age	No specific age	30-40 Y
- Smoking	↑↑ incidence in smokers	↓↓ incidence in smokers (80% non smokers)
- Abdominal Pain:	Prominent	Less prominent

	Crohn's disease	Ulcerative colitis
- Diarrhea	Yes	Yes <i>(severe & sometimes dysentery)</i>
Bleeding per rectum	Uncommon	Common
Anal & oral lesions	Yes	No
Bowel obstruction	May be	No
Malabsorption syndrome	May be	No
Intestinal fistula	May be	No
B ₁₂ deficiency	May be	No
Carcinoma risk	Minor	High
Systemic (extra intestinal manifestations)	• Skeletal : Arthritis, • Skin:erythema nodosa, pyoderma gangrenosa 🎵 • Liver: fatty liver – sclerosing cholangitis • Lung : interstitial pulmonary fibrosis. • Eye: uveitis, iritis	
Investigation		
- Radiological :	- deep ulcers (<i>cobble stone</i>) - areas of narrowing in the ileum. - Skip lesion - Fistula & strictures.	- shortened colon , absence of hustra (<i>lead pipe sign</i>) - pseudo polyposis 15%.
- Colonoscopy & biopsy	- Show involvement of the entire bowel wall with granuloma formation.	- Limited to the mucosa & may have crypt abscesses.

Treatment:**Medical treatment :**

- 1- Sulfasalazine: reduce inflammation especially in ulcerative colitis.
1 gm / 6 h reduced to a maintenance dose of 2 gm/ day for 2 years after remission .
- 2- Steroid (prednisone) : 60mg/d. This is the main treatment for active disease
- 3- Immuno Suppressants: Azathioprine (*Imuran*).
- 4- Symptomatic :
 - Antibiotics for 2ry infection : Metronidazole & ciprofloxacin.
 - Anti diarrheal drugs.

Surgical treatment : (Indications)

- Failure of medical treatment.
- Severe complications

IRRITABLE BOWEL SYNDROME (IBS)**Definition:**

It's a functional disturbance of colonic motility with no organic cause .

Etiology :

unknown , psychological disturbance play a role.

Clinical picture:

long history with long free interval

- Recurrent pain :over any part of the colon (especially in left iliac fossa).
- The pain is ↑↑ by food & ↓↓ by defecation .
- There is constipation or diarrhea .
- Abdominal distension is common .
- Patient with IBS often complain of anxiety , depression & tension headache.

Investigations :

(to exclude common diagnosis)

- no +ve finding .

Treatment :

- ☺ Reassurance .
- ☺ Diet : high fiber diet & bran , Avoid coffee , tea , smoking .
- ☺ Mild sedative , antispasmodics .
- ☺ Tegaserod (*Zelmac*) one tablet / 12 h : activate serotonin type 4 receptors in GIT .

DYSPEPSIA

Definition:

Any abdominal discomfort related to meal e.g. heartburn , epigastric pain ,
distension , nausea,.....

Causes of dyspepsia :

- **GIT causes :** - reflux esophagitis - peptic ulcer - cholecystitis
 - pancreatic disorders - hepatic diseases.
- **Systemic diseases :** Renal failure , hypercalcemia.
- **Drugs :** NSAID , cortisone .
- **Functional dyspepsia :** it's a persistent dyspepsia with no organic cause
(–ve endoscopy & the patient is Helicobacter pylori – negative).

Diagnosis :

History analysis:

- **What type of discomfort does the patient feel ?** e.g. heartburn , distension...
- **Is this discomfort related to meal ?**
- **Time to meal :**
 - **During meal :** Esophageal cause : Reflux esophagitis.
 - **½ H after meal :** Gastric cause e.g. Peptic ulcer .
 - **2-3 H after meal :** Duodenal cause e.g. Duodenal ulcer.
 - **8 H after meal :** Intestinal cause : obstruction .
- **Type of meal :**
 - ☺ **Fat :** Cholecystitis ☺ **Meat :** Cancer stomach . ☺ **Starch :** Gastric ulcer .
- **Associated symptoms .**

Investigations : for the cause

Patients with persistent dyspepsia need to be referred for **endoscopy** if they are **over 50 years old** , or they have associated weight loss , dysphagia , GIT bleeding or previous gastric surgery .

Treatment :

- ☺ **Diet** : eat little & often , Avoid fatty food, tea, coffee , smoking, alcohol.
- ☺ **Symptomatic treatment** .
- ☺ **Treatment of the cause** .

Diseases of Esophagus

- The esophagus is 25 cm long connecting the pharynx to the stomach .
- The mucosa is lined with squamous epithelium .
- The esophagus is protected from acid damage by a number of defenses including the lower esophageal sphincter pressure , gravity ,salivary bicarbonate & esophageal bicarbonate secretion .

Hiatus Hernia

- Occurs when part of the upper stomach herniates through the diaphragm into the chest .
- It's extremely common , especially with increasing age & obesity .
- **There are 2 types :**

1- Sliding 80 % :

- May be asymptomatic .
- May cause acid reflux , aspiration. - Bleeding may occur.
- **Investigations :** Barium swallow . upper endoscopy .
- **Treatment :**
 - Weight reduction (Meals should be small)
 - Sleeping in semi-sitting position .
 - H₂ blockers & proton pump inhibitors (*omeprazole*)
 - Prokinetic drug : domperidone (motilium).
 - **Surgery :** indicated in resistant cases (*fundoplication*)

2- Rolling 20 % : (para-esophageal hernia)

- Here , a small part of the fundus of the stomach rolls up alongside the esophagus through the hiatus .
- The gastro-esophageal junction is not affected so , there is no reflux
- may obstruct or strangulate .
- A big hernia may lead to mediastinal syndrome .
- Surgery is indicated in severe cases .

Esophageal Achalasia

Definition : It's a condition of unknown etiology resulting in abnormal peristalsis and lack of relaxation of the lower esophageal sphincter (LES) .

Clinical picture :

- Intermittent dysphagia : to fluid & to solid food .
- Regurgitation , chest pain , infection , mild weight loss may occur.

Investigation :

Barium swallow:-Esophageal dilatation with a smooth distal (bird's beak).
-Absence of gases in the fundus of the stomach.

Upper endoscopy .

Manometry : - high pressure in lower segment .
- Loss of peristalsis .

Treatment : Endoscopic dilatation or surgical myotomy .

Gastro-Esophageal Reflux Disease

(GERD)

Definition : Reflux of gastric contents into the esophagus.

Etiology & pathogenesis : (Failure of anti reflux mechanisms)

- 1- Decrease tone of lower esophageal sphincter (LES) .
- 2- Decrease esophageal clearance of acid due to poor esophageal peristalsis.
- 3- Delayed gastric emptying .
- 4- Hiatus hernia may aggravate the condition .

Predisposing factors :

- ◆ obesity . ◆ smoking . ◆ alcohol . ◆ coffee .
- ◆ large meals (especially late at night) .
- ◆ drugs : Ca antagonist , theophylline , anticholinergic & nitrates .

Clinical picture :

- 1- Heartburn : the main symptom .
- 2- Chest pain : - Epigastric & retrosternal (simulating angina) ☹️
- Increased on lying down & relieved by antacid .
- 3- Odynophagia : painful swallowing .
- 4- Dysphagia : due to disturbed motility .
- 5- Pulmonary : cough , asthma , aspiration pneumonia may occur .
- 6- Hematemesis & melena : due to mucosal inflammation or ulceration.
- 7- Laryngeal irritation .
- 8- **Barrett's esophagus** : (May occur in long standing acid reflux) .
- The normal squamous epithelium is replaced by the columnar gastric epithelium
- It's a premalignant leading to adenocarcinoma .

Investigations: (GERD is a clinical diagnosis)

- Esophageal PH monitoring (gold standard for diagnosis) . MCQ
- Endoscopy. - Manometry. - Barium swallow : it may show hiatus hernia .

Treatment :**1- Diet :**

- ☺️ Limit intake of food & drink that reduce lower esophageal sphincter pressure : fatty food , acidic foods , onions ,chocolate, coffee , alcohol .
- ☺️ Avoid heavy meals especially before sleep . ☺️ Weight reduction . ☺️ Stop smoking .

2- Postural therapy :

- ☺️ Raising the head of the bed at night .
- ☺️ Avoid lying down after eating .
- ☺️ Remain upright at least 2h after eating .

3- Drug therapy :

- ☺️ ↓ gastric acidity :
- Proton pump inhibitors : Omeprazole , most potent single agent.
- Antacid . - H₂ blockers : Ranitidine .
- ☺️ ↑ esophageal peristalsis : Domperidone (motilium)

4- Surgical & Endoscopic therapy : In resistant cases .

DYSPHAGIA

Definition: Difficulty in swallowing ,with a sensation of sticking or obstruction of the passage of food through the mouth , pharynx or the esophagus .

Causes :

1- Transfer dysphagia :

(alteration of neuromotor mechanism of the oropharyngeal phase, neurological causes)

- ☐ Bulbar palsy .
- ☐ Myasthenia gravis .
- ☐ Stroke .

2- Transit dysphagia : (motor disorders , abnormal peristalsis)

- ☐ Achalasia .
- ☐ Scleroderma .
- ☐ Esophageal spasm .

3-Obstructive dysphagia: (mechanical narrowing of the esophagus)

- ☐ Esophageal stricture .
- ☐ Cancer esophagus .
- ☐ All causes of mediastinal syndrome : Goitre , bronchogenic carcinoma , LN

4- Odynophagia : (painful swallowing)

- ☐ Inflammation of (mouth, tongue, pharynx, tonsils or esophagus)

5- Hysterical : (Globus hystericus)

Diagnosis of a case of dysphagia :

History analysis : Ask about :

- Onset , Course , Duration.
- Type of food (solid or liquid)
- Painless or painful .
- Associated symptoms .
- Site of obstruction .

Examples :

Transfer dysphagia :

- ♥ **Onset** : Acute ♥ **Course** : variable .
- ♥ **Type of food** : more to **liquid** .
- ♥ **Associated symptoms**: Nasal regurgitation , Dysphonia, neurological manifestations.

Transit dysphagia :

e.g. **achalasia**

- ♥ Intermittent & may progress –long duration.
- ♥ **Liquid** > solid.
- ♥ Relieved by repeated swallowing.
- ♥ **Associated symptoms** : Heartburn precipitated by eating , no marked loss of weight .

e.g. **scleroderma**

- ♥ Intermittent course .
- ♥ Both solids & liquids .
- ♥ Associated symptoms : Nocturnal cough , regurgitation .

NB : Dysphagia that worsens on ingestion cold liquids & improves with warm liquids suggests a motor disorder(transit dysphagia).

Obstructive dysphagia :

e.g. **cancer esophagus**

- ♥ Progressive – short duration .
- ♥ Dysphagia to **solids** that may progress to include liquids.
- ♥ Associated symptoms : weight loss , chest & back pain .

Investigations:

- ♥ **Barium swallow**: (*initial diagnostic step after history & examination*)
- ♥ Endoscopy .

Stomach & duodenum

Physiological aspect :

- HCL is secreted by the parietal cells of the stomach through the action of hydrogen-Potassium ATPase (proton pump)
- **Function of HCL :**
 - Converting pepsinogen to pepsin , initiating the first stages of protein digestion.
 - Antibacterial barrier.
- **HCL secretion is stimulated by :**
 - Vagus (directly & via increasing gastrin)
 - Gastrin.
 - Histamine.
- **HCL secretion is inhibited by :** **MCQ**
 - Somatostatin
 - Secretin
 - Cholecystokinin

N.B.

- Parietal cells → secret HCL & intrinsic factor.
- Peptic cells (chief) → secret Pepsin.
- G cells → secret Gastrin.

Peptic ulcer

Definition :

- Ulceration of mucosa which is exposed to acid peptic juice.
- The following sites are affected :
 - Duodenum.
 - Stomach.
 - Jejunum (after gastrojejunostomy)
 - Esophagus.
 - Meckel's diverticulum (contains ectopic gastric tissue)

Etiology :

It occurs when stomach acid penetrates the stomach and/or duodenal lining.

- It's a mix of :

- Hyperacidity : duodenal ulcer.
- Decreased mucosal resistance : gastric ulcer.

1- **Helicobacter pylori infection** (duodenal > gastric)

2- **Traditional NSAIDs :** (gastric > duodenal).

3- Rare causes : Zollinger Ellison syndrome (↑ acid production)

- **S**moking , **S**picy , **S**tress are no longer thought to be a cause of ulcers , but they can make ulcers worse (*aggravating factors*)

- Smoking → ↑ acid & ↓ protective prostaglandin.

1- Helicobacter pylori :

- Responsible for the majority of peptic ulcer (90% of DU , 70% of GU).
- H.pylori is a gram -ve spiral bacillus transmitted by feco-oral or through mouth to mouth such as kissing.
- Many people have H.pylori infection, but not everyone who has an infection will develop a peptic ulcer.
- Pathogenesis of H.pylori :
 - ↓ somatostatin.
 - ↑ gastrin release.
 - Produce cytotoxins causing mucosal inflammation.

2- Non steroidal anti-inflammatory drugs (NSAIDs) :

- NSAIDs act by inhibiting cyclo-oxygenase enzyme (COX) leading to decrease prostaglandin → mucosal erosions & ulceration.
- Notice that prostaglandin play a big role in gastric protection.

NB : All people at high risk of developing peptic ulcer should use COX-2 inhibitor rather than one of the traditional NSAIDs (COX-1 inhibitor) , however studies have shown that COX-2 inhibitors appear to increase the risk of heart attacks & stroke with long term use , so most doctors now use a traditional NSAIDs plus a strong acid inhibitor.

Disorder associated with peptic ulcer :

- **C**irrhosis.
- **C**OPD.
- **C**RF.
- **C**oronary diseases.

NB : *Peptic ulcer is more common in cirrhotics due to ↓ destruction of gastrin & histamine by the liver & ↓ mucosal resistance due to congestive gastropathy caused by portal hypertension.*

Clinical picture :

Symptom : Epigastric pain

- **Character :** burning , gnawing or dull ache.

- **Site :**

- DU : above the umbilicus & to the right of the midline.
- GU : epigastric & in the midline.

Sudden onset of severe generalized pain may indicate perforation.

- **Duration :** variable from few minutes to several hours.

- **Relation to meal :**

- DU : 2 - 3 h after meals.
- GU : precipitated by food $\frac{1}{2}$ h after meals

So, in DU : pain awakes the patient from sleep (*the most important symptom*)

- **Reliving factors :**

- DU : antacid or food , so appetite ↑ (patient eats frequently to relief pain)
- GU : fasting , vomiting (some patients learn to induce vomiting for pain relief)

- **Associated symptoms :**

anorexia , vomiting & weight loss in GU.

Signs : may be -ve

- **Localized tenderness** at the site of ulcer (**severe tender suggests a perforation**)
- Physical examination is critically important for discovering evidence of ulcer complications.

Complications :

- GIT **bleeding** : most common complication (15%) , **may be the first symptom.**
- **P**erforation : The second most common complication.
- **P**yloric obstruction.

Investigations :

- **Endoscopy** : The best to detect the ulcer.

- *Endoscopic biopsy should be taken in all gastric ulcer to differentiate between benign & malignant ulcer.*
- *Malignant gastric ulcer is mostly malignant from the start.*

- Occult blood in stool.
- Investigations for H. Pylori : antibodies , culture & sensitivity test.

Treatment :**i. Diet :**

- Frequent small meals.
- Avoid spicy food , smoking , alcohol.

ii. Medical treatment :**1. Antacid :** (*symptomatic relief in mild cases*)

e.g. mixture of Aluminum & Mg hydroxide (*Maalox*)

2. H₂ blockers : (for 4-8 weeks)

They block H₂ receptors → reduction of acid secretion.

- ✎ Cimetidine : 200 mg four times/d . SE : reversible gynecomastia & impotence.
- ✎ **Ranitidine** (*Zantac*) : 150 mg twice /d or better 300 mg at bed time.
- ✎ Famotidine : 20 - 40 mg/d.

3. Proton pump inhibitors (PPI) : (for 4 - 6 weeks)

- They inhibit H - K ATPase enzyme (proton pump) → ↓ H⁺ secretion → ↓ HCL secretion.
- ✎ Omeprazole (*Losec*) : 20 mg / d.
- ✎ Lansoprazole : 30 mg / d.
- ✎ Pantoprazole : 40 mg / d.

4. **Sucralfate:** (for 4 -6 weeks)

- It's a mucosal protective agent (*coating agent*)
- Dose : 1 gm / qid.
- Not given with antacid.

5. **Anticholinergic drugs:**

Pirenzepine (*gastrozepine*) : selective M₁ blocker.

6. **Eradication of H. Pylori:** (*triple therapy for 2 weeks*)

- No single agent is effective in eradication this organism.
- Triple therapy is used for 2 weeks **e.g.**
 - ✎ Omeprazole 20 mg twice daily.
 - ✎ Clarithromycin (*Klacid*) 500 mg twice daily.
 - ✎ Metronidazole 500 mg/d or Amoxycilline 1 gm /d twice daily.
- Eradication of H. Pylori may lead to dramatic decrease in ulcer recurrence to less than 5%.

7. **Avoid NSAIDs:**

If NSAIDs must be continued , either use selective COX-2 inhibitors as Lomoxicam (*Xefo*) or use traditional NSAIDs plus a strong acid inhibitor.

iii. Surgical treatment :

○ **Indication :**

Recurrent bleeding , Perforation , Pyloric obstruction , Malignancy.

○ **Operations :** Partial gastrectomy , Vagotomy.

iv. Treatment of complications :

○ **Bleeding :**

- Ranitidine or Omeprazole infusion.
- Endoscopic injection of adrenaline.
- Blood transfusion.

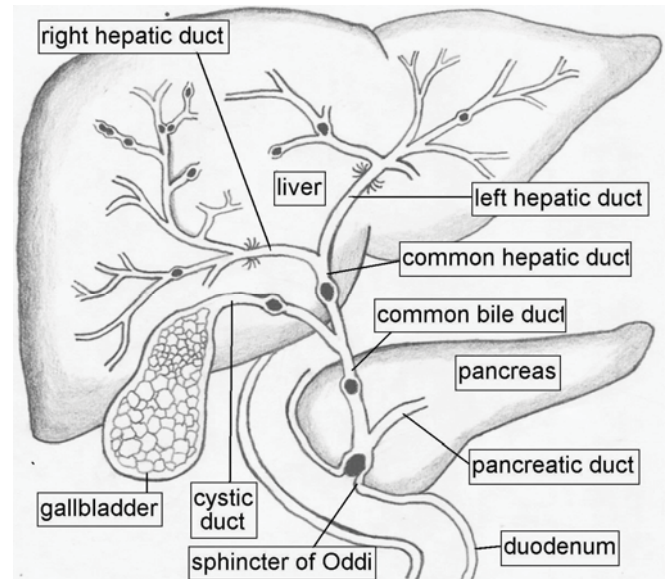
○ **Perforation :** IV fluid , analgesic & antibiotic then surgery.

○ **Pyloric obstruction :** surgery.

Gall bladder & Biliary system

Anatomy of Biliary system :

- The right & left hepatic duct arise from the right & left lobes of the liver & unite to form the common hepatic duct which is joined by the cystic duct from the gall bladder to form the common bile duct (CBD) which enters the duodenum (*often after joining the main pancreatic duct*) through the ampulla of Vater.
- The function of the gall bladder is to store bile that is produced in the liver before the bile is secreted into the intestines.
- Approximately 1 litre of bile is secreted by the hepatocytes each day .Half of this drain directly into the duodenum & the remainder is stored in the gallbladder . Cholecystokinin then stimulates its release.



Gall stones

Etiology :

1) Cholesterol stones :

Cholesterol is kept soluble by the presence of bile salts so , if bile salts decrease or cholesterol increases , cholesterol stones will occur.

2) Pigment stones : rare

Excessive production of bile pigments in bile e.g. chronic hemolysis.

80% gallstones are a mixture of cholesterol & bile pigment (*mixed stones*)

3) Stasis :

- Estrogen : due to GB wall relaxation.
- Long term parenteral nutrition : lack of oral intake → ↓ cholecystokinin that stimulate the GB contraction.

Risk factors for stone formation :

- Fatty (obesity) - Forty (increasing age) - Fertile (multi parity) - Female.
- DM - Drugs : contraceptive pills , Octreotide.

NB : Stones may form in the CBD even after cholecystectomy.

Clinical picture : gall stones are present in 10 - 20% of population

- **A**symptomatic : 80% of cases.
- **A**scending cholangitis (*bacterial infection*)
- **P**ancreatitis.
- **B**iliary colic.
- **C**holecystitis.
- **O**bstructive jaundice if duct obstruction.

Investigations :

- **US** : should be performed routinely in patients suspected of having gallstone disease
- **ERCP** : is the best technique for diagnosing common bile duct stones, but it is far less sensitive in detecting stones in the gallbladder than US.

NB : The major component of most gallstones is cholesterol, which is not radio-opaque. This explains why many gallstones do not show up on a plain X-ray; only stones with high calcium content are radio-opaque and visible.

Treatment :

- **Cholecystectomy is a definitive treatment** (*either surgical or by laparoscope*)
- Shock wave lithotripsy.
- Medical treatment : through the oral administration of bile acids, such as *ursodeoxycholic acid* to dissolve cholesterol stones.

Acute cholecystitis

Etiology & pathophysiology :

- It's usually caused by obstruction of the cystic duct by stones.
- Begins with sterile inflammation , then becomes infected (*E.coli* , *Strept. fecalis*)
- GB may distended with mucous (*catarrhal cholecystitis*) or pus (*suppurative cholecystitis*) .
- Rarely, acute gangrenous cholecystitis may develop.

Symptoms : **Severe pain**

- This pain starts usually in the right upper quadrant or epigastrium.
- May radiate to the back or right shoulder.
- Biliary pain typically is gradual in onset and lasts hours.
- Nausea & vomiting may occur.

Signs :

- Fever.
- Obstructive jaundice may occur due to edema or stone in CBD.
- Murphy's sign : ask the patient to take deep inspiration while palpating the G.B. area → the patient catch his breath due to sudden pain.
- Boas's sign : there is an area of hyperesthesia between the 9th and 11th ribs posteriorly on the right side.

Investigations :**1- Laboratory :**

- Leucocytosis.
- Serum bilirubin , AST , ALT & alkaline phosphates : may be elevated.

2- Radiological : US , Radioisotope scanning.**3- ECG :** done as a routine especially in the elderly patients to exclude IHD.**Treatment :****Medical :**

- Bed rest , IV fluid.
- Analgesics : pethidine (no morphine as it induces spasm of the sphincter of Oddi)
- Antibiotic : 3rd generation cephalosporin. ○ Antiemetic.

Surgical :

- If the attack subsides , cholecystectomy is done after 1- 3 months.
- Urgent surgery : in cases with complications.

Chronic Calculus Cholecystitis

Etiology : It's almost always associated with the presence of gall stones.

Clinical picture :

- There are recurrent attacks of right upper abdominal pain.
- Dyspepsia , local tenderness & Murphy's sign is usually present.

Investigations : US shows thick G.B. wall & gall stones.

Treatment : Cholecystectomy.

Acute Pancreatitis

Etiology :

- gall stones → obstruction of pancreatic duct (*the most common cause*)
- Alcohol.
- Viral infection e.g. mumps, Coxsackie B.
- Hyperlipidemia.
- Hypercalcemia.
- Idiopathic.

NB : Gall stones → back regurg of trypsin enzyme (*autodigestion*)

Symptoms :

- Abdominal Pain: epigastric pain radiating to the back.
- Nausea & vomiting.

Signs : *Unexpectedly, abdominal signs are minimal in comparison with the severity of pain.*

- Tenderness & rigidity : at first in the upper abdomen, later on become generalized.
- Cullen sign: umbilical ecchymosis.
- Grey turner's sign: ecchymosis in flanks.

Complications:

- Multiple organ failure (*MOF*) : HF, Renal failure, Respiratory failure.
- Abscess.
- Pancreatic ascites.
- Hypo Calcemia.
- DIC.
- Diabetes mellitus.
- Obstructive jaundice : due to edema of the head of the pancreas.

Poor prognostic indicators : *mnemonic : Pancreas*

- **P**aO₂ < 60mmHg .
- **A**ge > 55 years.
- **N**eutrophils: WCC > 16,000 /mm³.
- **C**alcium < 8mg %.
- **R**enal: Urea > 100mg %.
- **E**nzymes: LDH > 350 ; SGOT > 250 U.
- **A**lbumin < 3gm%
- **S**ugar: Glucose > 200 mg % .

Investigation:

1. Pancreatic enzymes :

- Amylase (N : 75-150 somogyi unit) : ↑↑ (in blood , urine , peritoneal fluid)
- Lipase : ↑↑ , more specific than amylase.

NB : *Serum amylase is raised in other conditions e.g. perforated peptic ulcer , ectopic pregnancy , alcoholics , salivary gland disease)*

2. Peritoneal aspiration : contains high amylase.

3. Blood picture : leucocytosis , anemia in hemorrhagic pancreatitis.

4. X ray, US, CT : gall stones , swollen pancreas , calcification.

5. ECG : to exclude myocardial infarction.

Treatment :

- Nasogastric suction.
- IV nutrition.
- Antibiotics.
- Analgesics : pethidine (no morphine as it induces spasm of the sphincter of Oddi)
- Somatostatin infusion.
- Treatment of complications e.g. Respiratory support , cortisone.
- Surgery for pancreatic abscess & ERCP may be needed.

Prognosis : death rate is about 10%.

Chronic Pancreatitis

Chronic pancreatitis is an inflammatory condition characterized by irreversible damage to the exocrine & later to the endocrine tissue of the pancreas.

Etiology : Like Acute pancreatitis but :

- **Chronic alcoholism** is the commonest cause.
- Gall stones are unlikely to cause chronic pancreatitis.

Clinical picture :

Triad of :

- i- Recurrent abdominal Pain: radiate to the back.
- ii- DM
- iii- Steatorrhea (*malabsorption* \$)

Investigation:

- Like acute pancreatitis.
- CT is the most sensitive for detection of pancreatic calcification.
- ERCP : shows irregular dilation and structuring of the pancreatic ducts.

Treatment :

1. Stop alcohol
2. Pancreatic enzyme replacement : taken as enteric coated tablets.
3. symptomatic treatment :
 - pain : Analgesics.
 - Treatment of DM.
 - Treatment of steatorrhea.

Cancer Head of Pancreas

- painless obstructive jaundice.
- loss of weight.
- Anorexia.

Cancer Body of Pancreas

- Pain: radiate to back.
- Jaundice: rare.

Tuberculous peritonitis

Etiology :

- **Primary :** Occurs in children by ingestion of infected milk (*bovine bacilli*)
- **Secondary :** Reactivation of a Tuberculous focus in the abdomen e.g. LN , Pulmonary TB.

Pathological type :

- 1- **Ascitic type :** Accumulation of free fluid in the peritoneum with no adhesion .
- 2- **Loculated type :** The peritoneal cavity is divided by fibrous adhesions into loculi containing ascetic fluid.
- 3- **Adhesive type :** Marked adhesion of the intestinal loops.

Clinical picture :

usually gradual

Symptoms :

- General symptoms : night fever, night sweating, loss of appetite, loss of weight.
- Constipation or diarrhea (*malabsorption syndrome*).
- Abdominal pain & distension.

Signs :

2 T , 2 P

- Toxemia : toxic face , fever ,....
- Tenderness with mild rigidity.
- Palpable masses (rolled omentum)
- Percussion may reveal shifting dullness.

Complications :

- ☛ Intestinal obstruction.
- ☛ Intestinal fistula.
- ☛ Spread of tuberculosis.
- ☛ Amyloidosis.

Investigations :

- i. Abdominal Ultrasonography : detect mild ascites , LN.
- ii. X ray : calcified mesenteric LN.
- iii. Aspiration of ascetic fluid :
 - Straw colored.
 - Rich in lymphocytes & proteins.
 - Z.N. or PCR for the organism.
- iv. Laparoscopy or Laparotomy.

Treatment :

Anti - Tuberculous drugs see chest

DD of epigastric pain :

Esophageal : - Esophageal spasm - Hiatus hernia. - Esophagitis. - Cancer.

Gastro-duodenal : - Gastritis - Peptic ulcer. - Gastric carcinoma.

Gall bladder : - cholecystitis. - Gall stones.

Pancreas : - pancreatitis. - Cancer pancreas.

Hepatic : - Hepatitis. - Congested liver. - Hepatoma.

Cardiovascular : -Angina - MI. - Pericarditis. - Aortic dissection.

Chest : -Pneumonia. -Pleurisy. - Repeated cough.

Nervous : - Herpes zoster. - Neurosis.

Medical conditions that mimic acute abdomen :

- Myocardial infarction.
- Rheumatic fever.
- Dissecting aortic aneurysm.
- Congestive heart failure : right upper abdominal pain due to congested liver.
- Diabetic ketoacidosis.
- Tetany.
- Hemolytic crisis.
- Henoch Schonlein purpura.
- Acute intermittent porphria.
- Acute pancreatitis.
- Renal failure.
- Hypoglycemia.
- Familial Mediterranean fever. (*FMF*)
- Vasculitis.
- Irritable bowel syndrome (*IBS*).

Acute pain in the upper abdomen

Oesophagitis	<i>Suggested by:</i> retrosternal pain, heartburn.
	<i>Confirmed by:</i> oesophagogastrosocopy.
Acute coronary syndrome (unstable angina or infarction)	<i>Suggested by:</i> chest tightness or pain on exertion.
	<i>Confirmed by:</i> exercise ECG , cardiac enzymes , coronary angiography .
Hiatus hernia	<i>Suggested by:</i> heartburn, worsens with stooping or lying, relieved by antacids.
	<i>Confirmed by:</i> oesophagogastrosocopy, barium meal.
Gastritis	<i>Suggested by:</i> epigastric pain, dull or burning discomfort, nocturnal pain
	<i>Confirmed by:</i> oesophagogastrosocopy, barium meal and pH study.
Gallstone colic (with no acute inflammation or infection)	<i>Suggested by:</i> jaundice, biliary colic, pain in epigastrium or right upper quadrant radiating to right lower scapula. No fever.
	<i>Confirmed by:</i> ultrasound of gallbladder and biliary ducts .
Acute cholecystitis	<i>Suggested by:</i> fever, guarding and positive Murphy's sign (abrupt stopping of inspiration when the palpating hand meets the inflamed gall bladder descending with the liver from behind the sub-costal margin on the right side, but not on the left side). ↑ WBC.
	<i>Confirmed by:</i> ultrasound gallbladder and biliary ducts .
Duodenal ulcer	<i>Suggested by:</i> epigastric pain, dull or burning discomfort, typically relieved by food, nocturnal pain.
	<i>Confirmed by:</i> oesophagogastrosocopy, barium meal and pH study: (<i>Helicobacter pylori</i> often present in mucosa or serology).
Gastric ulcer	<i>Suggested by:</i> epigastric pain, dull or burning discomfort, typically exacerbated by food, nocturnal pain.
	<i>Confirmed by:</i> oesophagogastrosocopy, barium meal and pH study.
Gastric carcinoma	<i>Suggested by:</i> marked anorexia, fullness, pain, large lymph node in the left supraclavicular fossa.
	<i>Confirmed by:</i> upper GI endoscopy with biopsy .
Pancreatitis	<i>Suggested by:</i> pain radiating straight through to the back, better on sitting up or leaning forward.
	<i>Confirmed by:</i> ↑ serum amylase , CT pancreas .

Acute central abdominal pain

Small bowel obstruction	<i>Suggested by:</i> vomiting, constipation with complete obstruction.
	<i>Confirmed by:</i> X ray shows small bowel loops and fluid levels.
Crohn's disease	<i>Suggested by:</i> chronic diarrhoea with abdominal pain, weight loss, palpable RLQ mass or fullness, mouth ulcers.
	<i>Confirmed by:</i> colonoscopy with biopsy, barium studies showing skip lesions.
Mesenteric artery occlusion	<i>Suggested by:</i> vomiting, bowel urgency, melaena, diarrhoea.
	<i>Confirmed by:</i> mesenteric angiography, exploratory laparotomy.
Abdominal aortic dissection	<i>Suggested by:</i> tearing pain , shock , hypotension and peripheral cyanosis.
	<i>Confirmed by:</i> ultrasound or CT abdomen.

Acute lateral abdominal pain

Pyelonephritis	<i>Suggested by:</i> pain in loin (upper lateral), rigors, fever, vomiting, frequency of micturition, renal angle tenderness.
	<i>Confirmed by:</i> CBC: leucocytosis. urinalysis : pyuria, urine culture and sensitivity
Renal calculus	<i>Suggested by:</i> renal colic mainly in loin (upper lateral), hematuria.
	<i>Confirmed by:</i> urinalysis, renal ultrasound, IVU, CT/MRI.
Ureteric calculus	<i>Suggested by:</i> renal colic, moving from loin (upper lateral) down to RLQ , hematuria.
	<i>Confirmed by:</i> urinalysis, renal ultrasound, IVU, CT/MRI.
Appendicitis	<i>Suggested by:</i> pain initially central, then radiating to <i>right</i> lower quadrant, anorexia, low grade fever, constipation. RLQ tenderness and guarding.
	<i>Confirmed by:</i> Inflamed appendix at laparotomy
Salpingitis	<i>Suggested by:</i> fever, nausea, vomiting, muco-purulent cervical discharge, irregular menses. Bilateral lower abdominal tenderness and guarding.
	<i>Confirmed by:</i> CBC: leucocytosis. High vaginal swab, laparoscopy.

Acute lower central (hypogastric) abdominal pain

Ulcerative colitis	<i>Suggested by:</i> abdominal pain, diarrhea with blood and mucus.
	<i>Confirmed by:</i> stool microscopy and culture, colonoscopy.
Large bowel obstruction	<i>Suggested by:</i> severe distension, late vomiting, visible peristalsis, resonant percussion, increased bowel sounds. Supine X ray showing peripheral abdominal large bowel shadow (with haustra partly crossing the lumen). Fluid levels on erect film.
	<i>Confirmed by:</i> abdominal ultrasound and laparotomy findings.
Cystitis	<i>Suggested by:</i> frequency, urgency, dysuria, hematuria.
	<i>Confirmed by:</i> urinalysis , culture & sensitivity test.
Ectopic pregnancy	<i>Suggested by:</i> constant unilateral pain , referred shoulder pain, amenorrhea , vaginal bleeding (usually less than normal period), faintness with an acute rupture.
	<i>Confirmed by:</i> pregnancy test +ve , bimanual examination reveals slightly enlarged uterus, pelvic ultrasound shows empty uterus.